

AD-A285 377



JMENTATION PAGE Dist: A

Form Approved  
OMB No. 0704-0188

It is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and reviewing the collection of information, sending comments regarding this burden estimate or any other aspect of this collection of information, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Rd to the Office of Management and Budget, Paperwork Reduction Project (0704-0188), Washington, DC 20543.

2. REPORT DATE  
19913. REPORT TYPE AND DATES COVERED 30 Sep 89-29 Jan  
Final Technical Report 1990-1991

|   |  |  |  |
|---|--|--|--|
| 4. TITLE AND SUBTITLE<br>Inspired Gas Composition Influences<br>Recovery from Experimental Venous Air Embolism  |  | 5. FUNDING NUMBERS<br>AFOSR-<br>89-0543<br>2312-AS   |  |
| 6. AUTHOR(S)<br>Joseph A. Bettencourt, M.D., Maj, MC,<br>Charles M. Harrison, M.D., Theodore Plemons, B.S., Sgt, USA<br>Patricia L. Schleiff, MS, William J. Mehm, Ph.D., Maj, USAF |  | 8. PERFORMING ORGANIZATION<br>REPORT NUMBER<br>AFIP/UBHG<br>AFOSR-TR- 94 0632                                |  |
| 7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)<br>Armed Forces Institute of Pathology<br>Division of Altitude and Hyperbaric Physiology<br>Washington, D.C. 20307               |  | 10. SPONSORING/MONITORING<br>AGENCY REPORT NUMBER<br>SELECTED<br>OCT 07, 1994                                |  |
| 9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)<br>Air Force Office of Scientific Research /NCL<br>110 Duncan Ave Suite 5115<br>Bolling AFB, DC 20332-5115<br>OF 1000000    |  | 12b. DISTRIBUTION CODE   |  |
| 11. SUPPLEMENTARY NOTES<br>None   |  | 12a. DISTRIBUTION/AVAILABILITY STATEMENT<br>DOD Approved for public release:<br>distribution is unlimited. A |  |
| 13. ABSTRACT (Maximum 200 words)<br><br>SEE ATTACHED<br><br>This document has been approved<br>for public release and sale; its<br>distribution is unlimited.                       |  |  |  |
| 14. SUBJECT TERMS<br>Embolicism, Air, Nitro s Oxide   |  | 15. NUMBER OF PAGES  |  |
| 17. SECURITY CLASSIFICATION<br>OF REPORT<br>Unclassified  |  | 16. PRICE CODE   |  |
| 18. SECURITY CLASSIFICATION<br>OF THIS PAGE<br>SAR  |  | 19. SECURITY CLASSIFICATION<br>OF ABSTRACT<br>SAR  |  |
| 20. LIMITATION OF ABSTRACT<br>SAR   |  | 21. LIMITATION OF ABSTRACT<br>SAR  |  |

NSN 7540-01-280-5500

Standard Form 298 (Rev. 2-89)  
Prescribed by ANSI Std. Z39-18

27 JUL 1994

**Best  
Available  
Copy**

## Introduction

### 1. Objectives

This study examined the effect of varying the breathing gas mixture on recovery from an experimentally induced venous air embolism (VAE). The specific objectives of this study were as follows:

1. To assess the lungs ability to dissipate a second air embolism.
2. To compare treatment gas breathing to air breathing with regard to:
  - a. Maximum change in physiological variables
  - b. Length of time taken for the return to baseline of physiological variables
  - c. Amount of residual intravascular air
  - d. Frequency with which venous air emboli are passed to the arterial circulation.

It was the intention of the original protocol to induce a venous air embolism for 30 minutes, allow the subject to recover for thirty minutes, ~~repeat the embolization for 30 minutes, again~~ allow the subject to recover for 30 minutes and then initiate a nitrous oxide challenge to evaluate for residual intravascular air. Following the initial embolization, it was planned to vary the composition of the breathing gas to: 1) room air, 2) pure O<sub>2</sub> at 1.0 atmospheres, 3) Sulfahexafluride 70% and oxygen 30%, 4) or pure oxygen at 2.4 atmospheres. After the final embolization, the breathing gas was to be switched to nitrous oxide (N<sub>2</sub>O) 70% and oxygen 30%.

Multiple technical preps were performed prior to actual data gathering. Initially, a goat model was developed. Much of the past animal work done in experimental VAE has been done in dogs, so we elected to change to a dog model, since the literature has shown it to be a valid, reliable model. During the technical prep phase, it was noted that the physiological variables that we monitored deviated from baseline early, then plateaued. Therefore, the protocol was refined to consist of one 15 minute air embolization period followed by a 30 minute recovery period on various breathing gas mixtures and the N<sub>2</sub>O challenge as a provocative test for residual air.

In addition, it was the intention of the original protocol to evaluate recovery from experimental VAE on oxygen at hyperbaric pressure (2.4 ATM). Numerous difficulties were encountered in attempting to interface with the hyperbaric chamber. Usable information could not be obtained from the pressure transducers at hyperbaric pressure and gases dissolved in blood at hyperbaric pressure quickly came out of solution prior to being analyzed at 1 ATM. A significant superiority had been noted in the group recovered on 100% oxygen, therefore, we elected to delete the group recovered on hyperbaric oxygen.

### 2) Findings

A cumulative, substantive and comprehensive statement and discussion of research background, rationale, material, methods and scientific significance can be found in the appendices. The major findings of this study are summarized as follows:

1) At the termination of the embolization periods, the end tidal carbon dioxide (ETCO<sub>2</sub>), which is an extremely sensitive indicator of VAE, rapidly returns to baseline. This rapid return to baseline of ETCO<sub>2</sub> suggests rapid recovery and pulmonary dissipation of the intravascular air. The return to baseline of ETCO<sub>2</sub> was noticeably faster in the group recovered on sulfahexafluride (SF<sub>6</sub>), although this difference was not statistically significant.

2) During the recovery period, the ETCO<sub>2</sub> of all three groups appears to plateau early.

3) At the initiation of the N<sub>2</sub>O challenge, the ETCO<sub>2</sub> of the SF<sub>6</sub> and air recovered groups decreased precipitously. The rapid decrease in the ETCO<sub>2</sub> in the air and SF<sub>6</sub> group indicates that, although the sensitive indicator of intravascular air had returned to baseline (ETCO<sub>2</sub>), pulmonary physiology remained disrupted from the initial insult.

4) Pulmonary injury following VAE persists after the physiological variables measured return to baseline. This pulmonary injury appears to be significantly attenuated by breathing 100% oxygen during the recovery period.

### 3) Presentations and Publications

21 June, 1991 - Publication of Abstract (Appendix A) and slide presentation delivered before The Undersea and Hyperbaric Medical Society Annual Meeting, June, 1991, San Diego, Ca.

Abstract citation: Bettencourt, J.A., Harrison, C.M., Plemons, T.J., and Mehm, W.J. "Oxygen Breathing Alters Response To Nitrous Oxide Challenge After Experimental Venous Air Embolism" Undersea Biomedical Research 1991

Manuscript in Preparation: Bettencourt, J.A., Harrison, C.M., Plemons, T.J., Schleiff, P.L., and Mehm, W.J. Inspired Gas Composition Influences Recovery From Experimental Venous Air Embolism To be submitted to: Journal of Neurosurgical Anesthesiology

### 4) Appendices

Appendix A - Abstract that accompanied a slide presentation delivered to the UHMS Annual Meeting in June 1991

Appendix B - Draft of research paper to be submitted to the Journal of Neurosurgical Anesthesiology

Indicate below the numbers and topics of sessions in which your abstract might be programmed (see list of suggested topics.)

1st Choice: # 4 Title Gas Physiology  
2nd Choice: # 1 Title Decompression Sickness

PRESENTATION PREFERENCE (scientific sessions only)  
Preferred choice (CHECK ONE ONLY)

☒ Slide presentation ☐ Poster presentation  
☐ Either

Do not fold this sheet  
Use cardboard backing  
when mailing

1991 UNDERSEA AND HYPERBARIC MEDICAL SOCIETY  
ANNUAL SCIENTIFIC MEETING ABSTRACT FORM

**OXYGEN BREATHING ALTERS RESPONSE TO NITROUS OXIDE CHALLENGE AFTER EXPERIMENTAL VENOUS AIR EMBOLISM.** J. A. Bettencourt, C. M. Harrison, T. J. Plemons and W. J. Mehn. Division of Altitude and Hyperbaric Physiology, Armed Forces Institute of Pathology, Washington, D.C., 20306-6000

Although it is presumed that the dissipation of venous air embolism is enhanced by breathing 100% oxygen, the literature in this area is inconclusive. This study examines the pulmonary response to nitrous oxide challenge after recovery from venous air embolism in anesthetized dogs. Sixteen dogs were randomized to either an air-recovered group or an oxygen-recovered group. To model venous air embolism, air was infused into the superior vena cava through the proximal port of a pulmonary artery (PA) catheter at the rate of 0.15 ml/kg/min for 15 min. Following the embolization, the animals were ventilated with either room air (air-recovered group) or 100% oxygen (oxygen-recovered group) for 30 mins. Thereafter, each group was ventilated with 70% nitrous oxide and 30% oxygen for 30 mins. End-tidal carbon dioxide (ETCO<sub>2</sub>) and PA pressures were monitored continuously. Arterial blood gases were measured every 5 mins during embolization and recovery, and every 10 mins during nitrous oxide challenge. During air embolization, we noted decreased ETCO<sub>2</sub>, increased pulmonary artery pressures, increased PaCO<sub>2</sub>, and profound hypoxia in both groups. During the recovery period, the arterial oxygen tension (PaO<sub>2</sub>) of the animals breathing 100% oxygen increased four to five fold while the PaO<sub>2</sub> of the room air breathing group remained moderately hypoxic. The PaCO<sub>2</sub> of both groups remained elevated throughout the treatment period. Upon nitrous oxide challenge, the ETCO<sub>2</sub> of both groups diverged. The ETCO<sub>2</sub> of the air-recovered group decreased precipitously, indicating an increase in pulmonary physiologic dead space. The ETCO<sub>2</sub> of the oxygen-recovered group, however, increased toward the elevated PaCO<sub>2</sub> value, indicating decreased pulmonary physiologic dead space. In contrast, there was no difference between groups with respect to PaCO<sub>2</sub> and PA diastolic pressure during recovery and nitrous oxide challenge. Since an increase in bubble size should be reflected in an increase in pulmonary artery pressure, we conclude that the difference in ETCO<sub>2</sub> between groups is not a result of simple changes in bubble size, but may be a more complex response to hyperoxia in the recovery period.

Accession For

NTIS CRA&I ☒

DTIC TAB ☐

Unannounced ☐

Justification

By

Distribution/

Availability Codes

1st

Avail and/or  
Special

A-1

Name and mailing address of presenting author:

Joseph A. Bettencourt, CPT, MC, USA

AFIP (CPW-P)

Washington, DC Zip 20306-6000

Telephone No.: Area Code 202 # 576-2868

**IMPORTANT:** Your abstract will appear in the Program/Abstract book exactly as you submit it.

Read all instructions before you type abstract. Also see sample abstracts and typing instructions on reverse side.

The original ribbon copy of this abstract form (for reproduction by photo-offset) must be submitted together with, where possible, 10 photocopies.

SEE REVERSE FOR ABSTRACT DEADLINES  
AND MAILING INSTRUCTIONS

Inspired Gas Composition Influences Recovery From  
Experimental Venous Air Embolism

Joseph A. Bettencourt M.D., Cpt., M.C., U.S.A.

Charles M. Harrison M.D., Maj., M.C. U.S.A.R.

Thoedore Plemons B.S., Sgt., U.S.A.

Patricia L. Schleiff, M.S.

William J. Mehm Ph.D., Maj., U.S.A.F.

Armed Forces Institute of Pathology, Division of Altitude  
and Hyperbaric Physiology, Washington, D.C. 20307

Send correspondence to:

Joseph A. Bettencourt M.D., Cpt., M.C.

Anesthesiology Department

Walter Reed Army Medical Center

Washington, D.C.

Phone Number: 202-576-1471

Running Head: Recovery From Venous Air Embolism

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army, the Department of the Air Force, or the Department of Defense.

### Abstract

Venous air embolism (VAE) is a potentially fatal occurrence frequently encountered in neurosurgical procedures performed in the sitting position. The morbidity of this event has been reduced primarily by efforts at early detection and prevention. Clinically, VAE is accompanied by hypoxia, hypercarbia, and an increase in dead space, manifested initially by a precipitous fall in end tidal carbon dioxide (ETCO<sub>2</sub>). Treatment consists of identifying and controlling the source, and hyperventilation on 100% oxygen. Hemodynamic support is given as required. A canine model of VAE was used to evaluate the effect of different inspired gas mixtures on the recovery from continuous venous air infusion. Sulfur hexafluoride (SF<sub>6</sub>), a non-hyperoxic, nitrogen free inspired gas was tested to determine if it would be a preferable alternative to recovery on 100% oxygen. Residual air effect was identified after the recovery period by a nitrous oxide challenge. In this study, recovery from VAE on 100% oxygen, as determined by response to nitrous oxide, was demonstrated to be significantly superior to either room air or SF<sub>6</sub>. ETCO<sub>2</sub>, pulmonary artery diastolic pressure (PAD) and arterial oxygen tension (PaO<sub>2</sub>) all demonstrated a greater ability to tolerate the nitrous oxide challenge in subjects recovered with 100% oxygen.

Key Words: Embolism, air, nitrous oxide, end tidal CO<sub>2</sub>, hyperoxia

## Introduction

Venous air embolism (VAE) poses a serious hazard to patients who undergo neurosurgical, craniofacial, and open-heart procedures. Typically, VAE causes a precipitous decrease in end tidal carbon dioxide (ETCO<sub>2</sub>) and arterial oxygen tension (PaO<sub>2</sub>), accompanied by an abrupt increase in arterial carbon dioxide tension (PaCO<sub>2</sub>) and pulmonary artery pressures. The standard intraoperative therapy for VAE is to prevent further entrainment of air and to dissipate the air embolus using ventilation with by 100% oxygen. Because 100% oxygen is nitrogen-free, ventilation with 100% oxygen is presumed to hasten resolution of VAE by creating a favorable alveolar gradient for excretion of nitrogen. However, few studies comparing oxygen and other nitrogen-free breathing gas mixtures have been done. Additionally, the mechanism by which oxygen enhances recovery from VAE may not be due to enhanced nitrogen excretion. For example, Russell et al. (1), after inducing VAE in dogs with <sup>15</sup>N<sub>2</sub>, found no significant difference in heavy nitrogen excretion among animals ventilated with room air, 100% oxygen, or hyperbaric oxygen (HBO). Lastly, although the resolution of VAE is clinically ascertained by return of ETCO<sub>2</sub>, pulmonary artery pressures, PaO<sub>2</sub>, and PaCO<sub>2</sub> to pre-embolus values, these findings do not guarantee total physical resolution of intravascular air. This prompted Shapiro and colleagues to develop the "nitrous oxide challenge" as a means of revealing residual intravascular air (2). In this study, we used a canine VAE model to compare the extent



of recovery attained by 100% oxygen ventilation versus 70% sulfur hexafluoride (SF6) (a dense, inert gas)/ 30% oxygen, a nitrogen free mixture. Our data indicate that although ETCO2 and pulmonary artery pressures returned to pre-embolism values most rapidly in the SF6/oxygen recovered group, the same variables deteriorated most rapidly in the SF6/oxygen recovered group on nitrous oxide challenge. This suggests that the resolution of VAE during 100% oxygen breathing is not explained entirely by the absence of nitrogen in the inspired gas.

### Materials and Methods

All experimental procedures were reviewed and approved by our institutional laboratory animal use committee. The experiments reported herein were conducted according to principles described in "Guide for the Care and Use Of Laboratory Animals" prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council, U.S. Department of Health and Human Services Publication No. (NIH) 85-23.

### General Preparation

Mongrel dogs of either sex weighing between 20 and 30 kg were fasted overnight and anesthetized with pentobarbital 20-30 mg/kg. After tracheal intubation, anesthesia was maintained with additional pentobarbital titrated to ablate ventilatory efforts by the animal. The lungs were mechanically ventilated on room air with a volume cycled ventilator (Harvard Apparatus, South Natick, MA.). The tidal volume was 10-12 ml/kg, and when the ETCO<sub>2</sub> reached between 35-45 mmHg., the ventilation was held constant. Following induction of anesthesia and intubation, a femoral artery was cannulated with a 20 gauge 2 inch catheter and the right external jugular vein was cannulated with an 8 french percutaneous introducer (PCI). When difficulty with percutaneous cannulation was encountered, the vessel was surgically exposed and directly cannulated. A pulmonary artery (PA) catheter was placed through

the PCI and correct positioning in the pulmonary artery was confirmed by waveform analysis. Pulmonary artery pressure and systolic and diastolic arterial blood pressure were monitored continuously using pressure transducers (Gould, Greenbelt, Md.). End tidal carbon dioxide (ETCO<sub>2</sub>) was monitored using a sidestream capnograph (Marquette Electronics, Milwaukee, WI). Arterial blood gases were analyzed using a Corning 170 pH/ Blood Gas Analyzer (Corning Medical, Corning Glass Works, Medfield, MA.).

### Experimental Protocol

Twenty-four animals were randomized to one of three experimental groups. The 3 groups were designated according to the composition of the gas inspired during the recovery period. Individual experiments were conducted in 3 periods: embolization, recovery, and nitrous oxide challenge (Fig. 1). All dogs were initially ventilated with room air. Baseline values for ETCO<sub>2</sub>, pulmonary artery diastolic pressure (PAD), systolic and diastolic blood pressure, and arterial blood gas (ABG) were obtained after the animals were allowed to stabilize for 30 minutes. Then, while ventilated with room air, each animal received a continuous infusion of air through the proximal port of the PA catheter at the rate of 0.15 ml.kg<sup>-1</sup>.min<sup>-1</sup>. PAD and ETCO<sub>2</sub> were recorded each minute. Arterial blood pressures and ABG were recorded every 5 minutes. Following the 15 minute air embolization period, the air infusion was terminated and each animal was switched to 1 of

3 inspired-gas mixtures: 1) room air 2) 100% oxygen or 3) 70% SF<sub>6</sub>/30% oxygen. The recovery period lasted for 30 minutes. Immediately following the recovery period, all animals were switched to 70% N<sub>2</sub>O/30% oxygen for 30 minutes. During the nitrous oxide challenge, ETCO<sub>2</sub> and PAD were again recorded every minute, arterial pressure every 5 minutes and ABG every 10 minutes. Immediately thereafter, each animal was euthanized with intravenous potassium chloride.

A non parametric statistical analysis, the Kruskal- Wallis, was performed at an alpha level of 0.05.

## Results

The data were analyzed as the mean difference in baseline values (time 0 value was subtracted from time x 's value). The 7 response variables were: 1) ETCO<sub>2</sub>, 2) PaCO<sub>2</sub>, 3) PaO<sub>2</sub>, 4) PAD, 5) systolic blood pressure (SYS), 6) diastolic blood pressure (DIA), and arterial pH. The treatment groups were divided based on the inspired gas composition during the recovery period. The statistical analyses were performed in three sections: time 0 to time 15, time 15 to time 45, and time 45 to 75. For the initial 15 minute time period, during the embolization, there were no significant differences in the mean baseline differences between the three treatment groups for the response variables PaCO<sub>2</sub>, PaO<sub>2</sub>, SYS, DIA, and pH. During the later portion of the embolization period, there were differences in the PAD (from time 11 to time 14). Figure 2 depicts the early divergence between ETCO<sub>2</sub> and PaCO<sub>2</sub> which is anticipated clinically and has been observed by others. At the termination of the embolization, the ETCO<sub>2</sub> rapidly returns to baseline, noticeably faster in the group recovered with SF<sub>6</sub> although this difference is not statistically different. In contrast to ETCO<sub>2</sub>, the PaCO<sub>2</sub> remains abnormal in the air and oxygen groups, however in the SF<sub>6</sub> group, PaCO<sub>2</sub> returns to normal. At times 20 and 25 minutes, early in the recovery period, there were significant differences in the value of PaCO<sub>2</sub> between SF<sub>6</sub> and oxygen and SF<sub>6</sub> and air. At time thirty, there was a significant difference between SF<sub>6</sub> and oxygen. During the recovery period, the ETCO<sub>2</sub> appears to plateau in all 3 groups. At the initiation of the

nitrous oxide challenge, the ETCO<sub>2</sub> of the SF<sub>6</sub> and air groups both decreased precipitously. As in the initial embolization period, the ETCO<sub>2</sub> rapidly decreased, indicating that the pulmonary physiology remained disrupted from the initial insult. The precipitous drop in ETCO<sub>2</sub> was even more remarkable in the SF<sub>6</sub> group than in the air recovered group. In the early nitrous oxide challenge, the ETCO<sub>2</sub> of all 3 groups was significantly different. There were significant differences between oxygen and SF<sub>6</sub> and air and SF<sub>6</sub> for times 46 to 50, 60 to 68 and 71 to 75. For times 51 to 59, 69 and 70, there were significant differences between all 3 groups (Fig. 2). PaO<sub>2</sub> was statistically different between all three groups during the recovery period, which can be attributed to the different inspired concentrations of oxygen. During the nitrous oxide challenge, at times 55, 65 and 75, the PaO<sub>2</sub> of the SF<sub>6</sub> group was significantly lower than either the air or oxygen recovered groups (Fig. 3). During the nitrous oxide challenge, the PAD of the SF<sub>6</sub> group increased, recapitulating its response during the initial embolization. There were significant differences in PAD between SF<sub>6</sub> and oxygen for times 52 and 57 in addition to differences between SF<sub>6</sub> and oxygen and SF<sub>6</sub> and air for times 53 to 56 and 59. There was a difference between SF<sub>6</sub> and air for times 60 and 61.(Fig.4). The data strongly suggest that pulmonary injury persists after the physiologic variables measured returned to baseline and that this injury is tempered by an inspired gas mixture of 100% oxygen.

## Discussion

The nitrous oxide challenge as described by Shapiro et al. (2) and Munson (3) is a technique used to identify residual venous air following VAE. Shapiro et al. (2) showed that following VAE, physiologic variables may return to baseline frequently after a brief recovery period. Russell et al. (1), using VAE induced by heavy nitrogen were able to detect the expired gas ( $^{15}\text{N}_2$ ) by mass spectrometry. They showed rapid pulmonary excretion of the VAE within 10 minutes, regardless of the inspired gas composition (room air, 100% oxygen, hyperbaric pressure or 100% oxygen at hyperbaric pressure). This strongly suggests that return of physiologic variables to baseline, although suggestive of dissipation of the VAE, cannot be assumed to indicate the resolution of the pulmonary effects of the VAE. Although, as Russell concluded (1), the inspired gas composition may not affect the physical removal of the air, the present study strongly indicates that the inspired gas composition following VAE influences the pulmonary response to the air insult. Sergysels et al. (4) utilized a dog model to further evaluate the contribution made by inspired gas composition on recovery from experimental VAE. They noted a rapid return of physiological variables to baseline following VAE and ventilation with  $\text{SF}_6$ , which was reversed with air and nitrous oxide breathing, and not seen with helium and oxygen breathing. Sergysels reports the only physiologic change associated with  $\text{SF}_6$  breathing alone is

THIS  
PAGE  
IS  
MISSING  
IN  
ORIGINAL  
DOCUMENT



of nitrogen, as Russell et al. did. Our results support those of Sergysels et al. demonstrating a rapid return to baseline of physiologic variables with SF6 breathing following VAE.(4)

Similarly, Butler et al., using a dog model of VAE, found that preoxygenation prior to air infusion improved the ability to dissipate air as detected by nitrous oxide challenge.(5) These findings of Butler and those of the present study support our premise that 100% oxygen in some way attenuates the damage to the lungs caused by VAE. These findings are not necessarily in conflict with those of Russell(1). Although they reported no influence on the dissipation of air by the inspired gas composition, they did not report evidence of pulmonary damage as evidenced by decreased function in gas exchange. Even though the air bubble may no longer be an actual physical entity, pulmonary function remains disrupted and this disruption is rendered less severe by ventilation with 100% oxygen.

Clinically, these findings support the use of 100% oxygen in the neurosurgical patient for sitting craniotomy. This practice would improve the individual patient's ability to tolerate VAE and also aid in the detection of VAE by observing for end tidal nitrogen by mass spectrometry.

### Acknowledgments

The authors wish to thank Tsgt. Joseph G. Casale, U.S.A.F., SSgt. Roderick F. Herring, U.S.A.F., SSgt. Bernard Wilson, U.S.A.F., SSgt. David A. Nelson, U.S.A.F., Maj. Darrell W. Criswell, U.S.A.F., Maj. Loraine H. Anderson, U.S.A.F., MSgt. Frank J. Roberts, U.S.A.F., and Kenneth Muvich, Ph.D. for technical assistance. We would also like to thank Maj. John A. Bley Jr., V.C., U.S.A., Cpt Rebecca Cockman-Thomas, V.C., U.S.A., Sgt. Eva Herrera-Lohr, U.S.A., and Spc. Alfonsa Rullan-Borrero, U.S.A. for assistance in care and preparation of the animals.

## References

- 1) Russell G, Snider M, Richard R, Loomis J: Venous air emboli with 15N2: pulmonary excretion and physiologic responses in dogs. Undersea Biomed Res 1991: 18: 37-45.
- 2) Shapiro H, Yoachim J, Marshall L: Nitrous oxide challenge for detection of residual intravascular pulmonary gas following venous air embolism. Anesth Analg 1982: 61 (3): 304-306.
- 3) Munson E: Pathophysiology and treatment of venous air embolism. M.E.J. Anesth 1988: 9:315-325.
- 4) Sergysels R, Jasper N, Delaunois L, Chang H, Martin R: Effect of ventilation with different gas mixtures on experimental lung air embolism. Respiration Physiology 1978: 34: 329-343.
- 5) Shirakusa T: Fatal pneumopericardium caused by SF6 gas infusion into the pleural space after pneumonectomy and pericardial resection. Chest 1989: 95 (6): 1350-1351
- 6) Meyer M, Schuster K, Schulz H: Alveolar slope and dead space of He and SF6 in dogs: comparison of airway and venous loading. Journal of Applied Physiology 1990: 69 (3): 937-944
- 7) Butler B, Luehr S, Katz J: Venous gas embolism: time course of residual pulmonary intravascular bubbles. Undersea Biomed Res 1989: 16: 21-29.

**Air infusion  
0.15 ml/kg/min**

**↓ 15 min**

**Recovery  
100% Oxygen**

**Room air**

**SF6/oxygen 70/30**

**↓ 30 min**

**Nitrous Oxide Challenge**

**30 min**

**Variables:**

**Pa CO<sub>2</sub>**

**PaO<sub>2</sub>**

**P.A.D.**

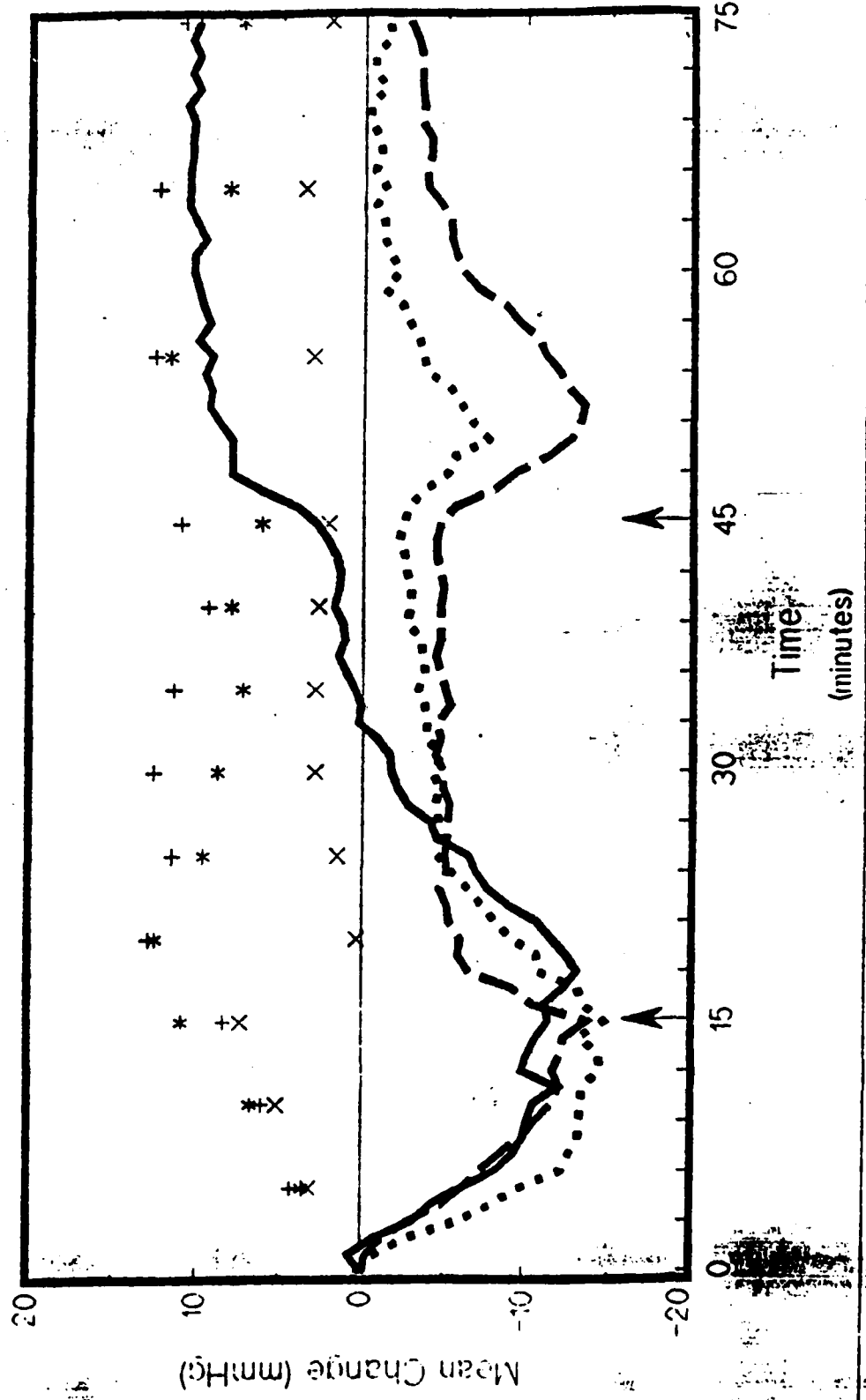
**ET CO<sub>2</sub>**

# Venous Air Embolism

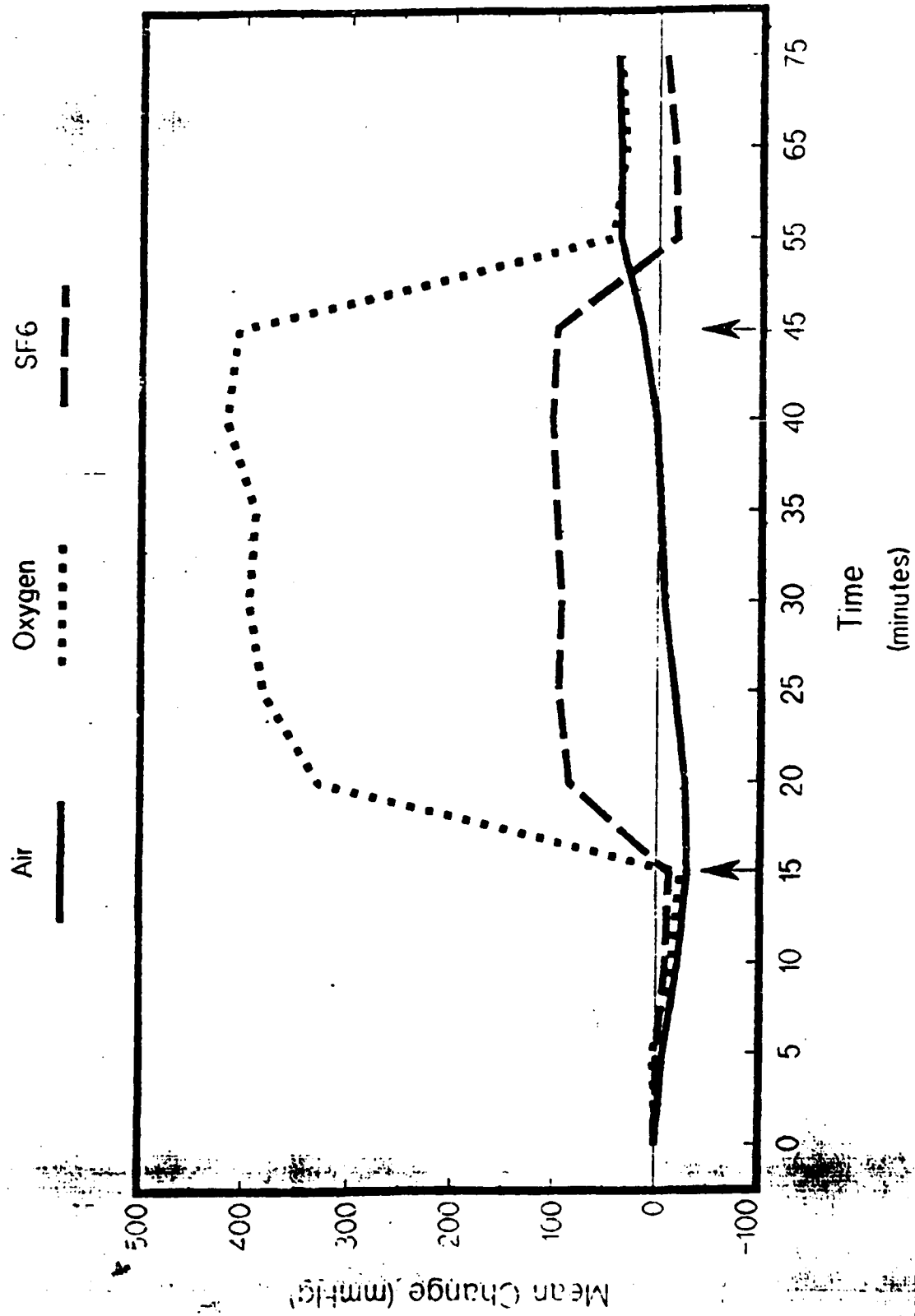
PaCO<sub>2</sub>/ETCO<sub>2</sub>

Air \* Oxygen + SF6 x

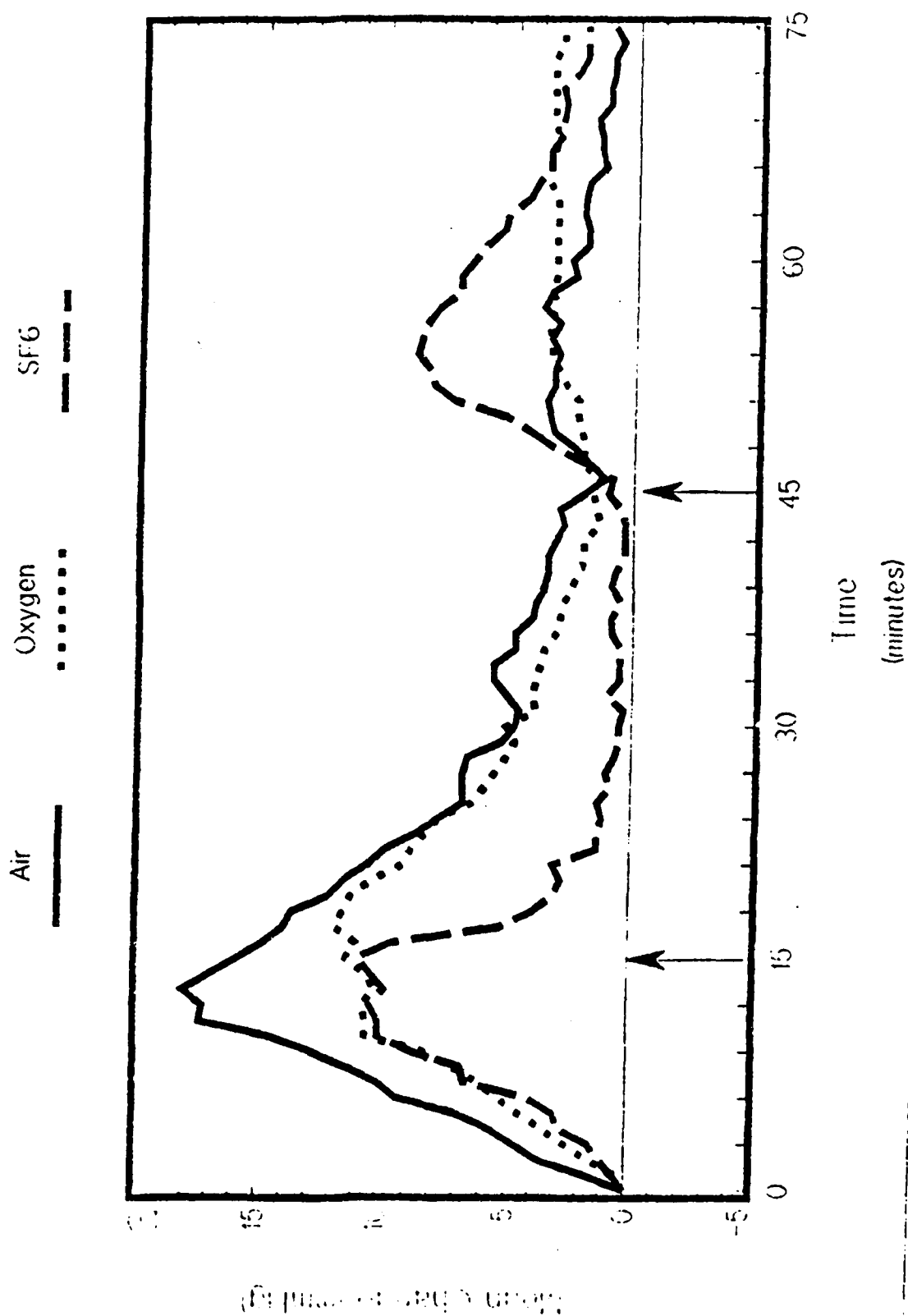
Air — Oxygen ... SF6 - - -



# Venous Air Embolism PaO2



# Venous Air Embolism Pulmonary Artery Diastolic Pressure



### Legends

Fig. 1. The experimental protocol consisted of venous air infusion at a rate of 0.15ml/kg/min for 15 min followed by recovery on 100% oxygen, room air or SF6 70% and oxygen 30%. The recovery period was followed by a nitrous oxide challenge (N2O 70% and O2 30%) for thirty min. The variables of interest were PaCO2, PaO2, P.A.D., and ETCO2.

Fig. 2. This depicts the mean difference from baseline values plotted against time for PaCO2 and ETCO2 for all 3 experimental groups. Note the rapid return to baseline of the PaCO2 of the SF6 group following the embolization. Also significant is the rapid decline in ETCO2 at the beginning of the nitrous oxide challenge.

Fig. 3. This depicts the P.A.D. for the 3 groups. The SF6 group is notable for a rapid return to baseline following the embolization period and a rapid increase from baseline at the beginning of the nitrous oxide challenge.

Fig. 4. This shows the mean difference from baseline in PaO2 for the 3 experimental groups. The wide variability during the recovery period is attributable to differences in inspired oxygen tension (FiO2). During the nitrous oxide challenge, the PaO2 of the SF6 group is significantly lower than the other groups, suggesting decreased pulmonary function exacerbated by nitrous oxide.